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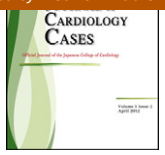

Case Report

Various morphologies of bidirectional ventricular tachycardia caused by aconite "Torikabuto" poisoning

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ABSTRACT

A 43-year-old man presented with nausea. The patient developed ventricular fibrillation (VF), which was refractory to antiarrhythmic drugs and defibrillation. A coronary angiogram showed no coronary artery stenosis. We recorded various fatal arrhythmias, including bidirectional ventricular tachycardia (BVT). The presence of multiple types of BVTs that were refractory to drugs such as adenosine triphosphate, isoproterenol, verapamil, propranolol, and pilsicainide, and easily recurred after defibrillation indicated aconite poisoning. After persisting for 24 h, VF spontaneously resolved and sinus rhythm was restored. Laboratory data revealed lethal concentrations of aconitine. To the best of our knowledge, this is the first report of aconite poisoning-induced BVTs manifesting with multiple morphologies on 12-lead electrocardiogram. The arrhythmogenic effects of aconitine are well recognized. In addition to causing VT and VF, aconitine also can cause BVT. Aconitine can lead to delayed afterdepolarization which has an important role in triggering and maintaining BVT. However, in this case, the concentration of aconitine was high enough to render these drugs ineffective. Prompt application of percutaneous cardio-pulmonary support, which was continued until the aconitine was metabolized, proved successful in this case and should be considered as a management approach in cases of severe aconite poisoning.

Introduction

Bidirectional ventricular tachycardia (BVT) is a rare but intriguing arrhythmia, which is characterized by an alternating beat-to-beat QRS complex on the 12 lead electrocardiogram (ECG). Clinically, BVT is commonly associated with digitalis toxicity, and has been observed in a clinical setting, such as catecholaminergic polymorphic VT (CPVT) and aconite poisoning. The mechanisms are considered to be delayed afterdepolarization and rarely reentry.

Case report

A 43-year-old man with no apparent medical history presented with nausea. On arrival at our department, he showed an altered level of consciousness and was agitated. The patient suddenly developed ventricular fibrillation (VF), which was refractory to antiarrhythmic drugs and defibrillation. He received percutaneous cardiopulmonary support (PCPS) to account for the possibility of acute coronary syndrome. However, a coronary angiogram showed no coronary artery stenosis. He underwent intraaortic balloon pumping (IABP) and continuous hemodiafiltration (CHDF) and was cared for in the coronary care unit; his VF had still not resolved at this point. We recorded various fatal arrhythmias in the coronary care unit. We recorded 6 types of BVT (Fig. 1A–F), among which the BVT alternating between the left bundle branch block and right bundle blanch block is considered to be rare (Fig. 2). BVT persisted for a few minutes, occasionally sustained for more than ten minutes after termination of VF by defibrillation. Intravenous infusion (IV) of various drugs such as adenosine triphosphate (20 mg, IV), isoproterenol (1 μg/min, continuous intravenous infusion [CIV]), verapamil (5 mg, IV), propranolol (10 mg by 10 min, CIV), and pilsicainide (1 mg/kg, by 10 min, CIV) were not effective for BVT, polymorphic VT, VF, and monomorphic VT. The drugs were administered in order of shorter metabolized time and there was enough

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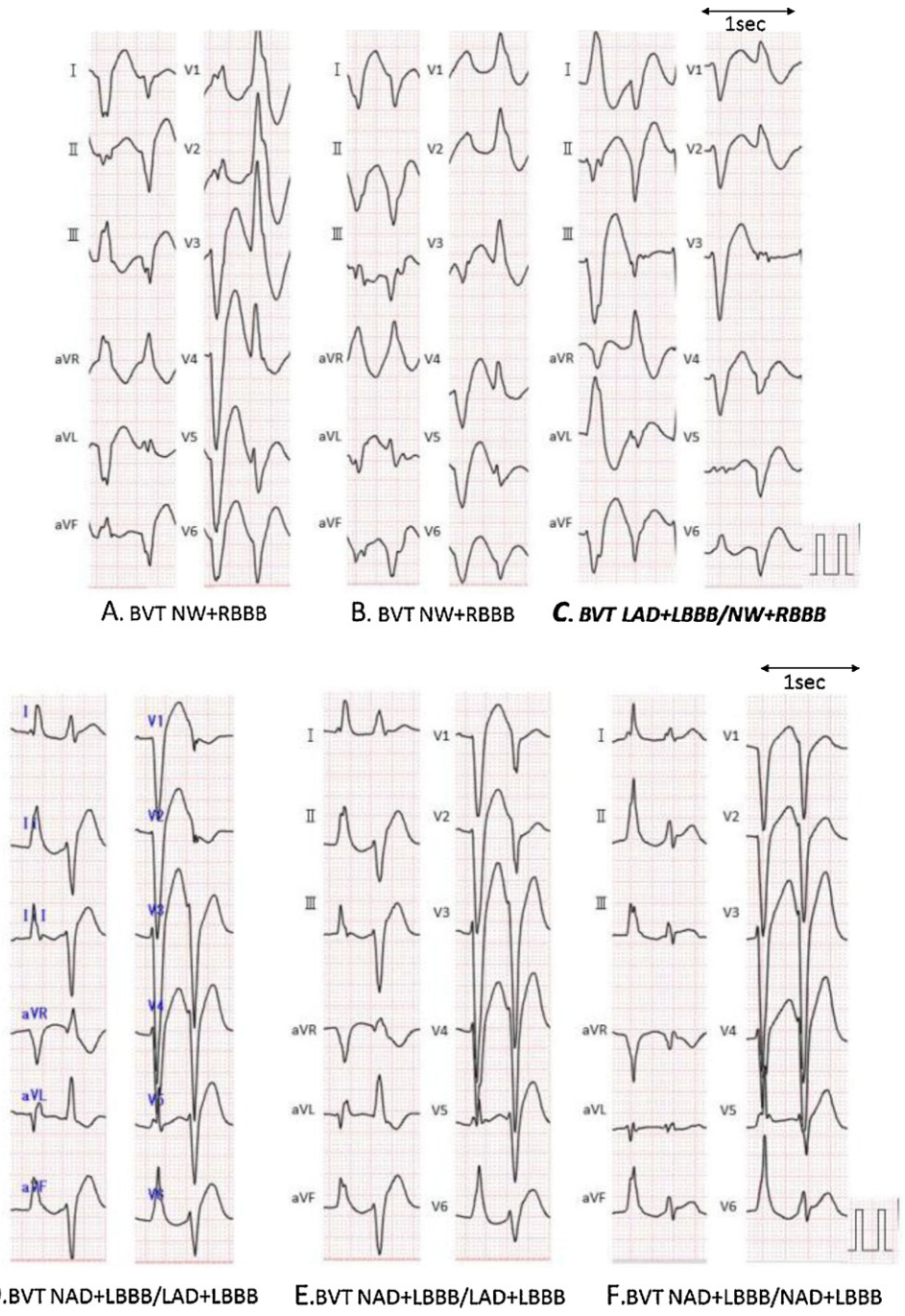

Fig. 1. (A–F) Presentation of 12-lead electrocardiograms of 6 configurations of BVT in this case. "C" is considered to be rare. BVT, bidirectional ventricular tachycardia; NW, north west axis deviation; LBBB, left bundle branch block; RBBB, right bundle branch block.

time between each drug administration to be metabolized. Some BVTs had spontaneously changed from one type to another type, however finally changing to polymorphic VT or VF. The presence of multiple types of BVT and fatal ventricular arrhythmias that were refractory to drugs and easily recurred after defibrillation indicated aconite poisoning. However, after persisting for 24 h, VF spontaneously resolved and sinus rhythm was restored. Laboratory data obtained at admission revealed lethal concentrations of aconitine in the serum (15.8 ng/mL); therefore, we diagnosed a case of aconite poisoning. The next day, about 12 h after admission, the data showed a lower value (10.3 ng/mL). The patient recovered well and achieved normal social functioning thereafter. He told us the reason why he was exposed to such a high concentration of aconitine. When he had taken edible wild plants as a pastime and stored them about 1 week before admission, he had not known them as "Torikabuto". Then a week later he ate them all in the morning as a breakfast, therefore, he developed symptoms of poisoning 3 h later.

Discussion

BVT is a rare but intriguing arrhythmia characterized by an alternating beat-to-beat QRS complex on the 12-lead ECG. BVT is typically associated with digitalis toxicity but has been observed in other clinical settings such as catecholaminergic polymorphic VT and aconite poisoning [1].

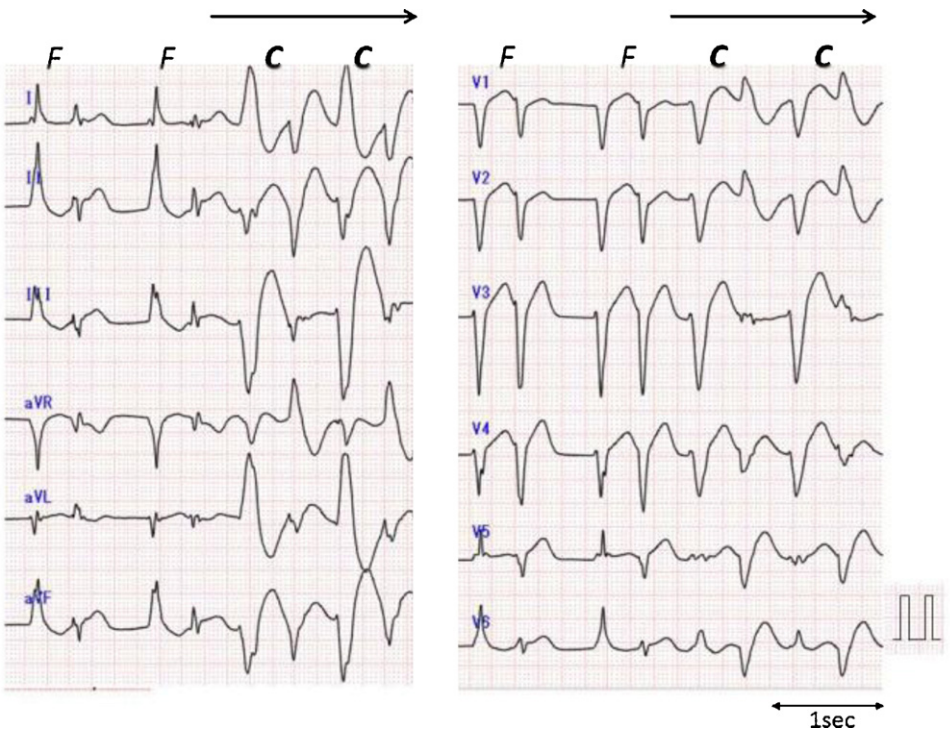

Fig. 2. Presentation of 12-lead electrocardiogram (ECG) of clinical bidirectional ventricular tachycardia (BVT). The morphology of BVT was drastically changing "F" to "C". The BVT alternating between the left bundle branch block and right bundle blanch block is considered to be rare. The ECG of BVT we diagnosed never showed sinus rhythm and narrow QRS complex.

To the best of our knowledge, there are no case reports of aconitine poisoning-induced BVT manifesting with multiple morphologies on 12-lead ECG. The arrhythmogenic effects of aconitine are well recognized. In addition to causing polymorphic VT and VF, aconitine can also cause BVT. Aconitine binds to the open state of the voltage-sensitive sodium channel and inhibits the inactivation of the channel. The consequent prolonged inward current of the sodium channel leads to intracellular accumulation of Na^+ and activates the Na–Ca exchanger, causing Ca^{2+} overload and delayed afterdepolarization (DAD) [2]. Several reports suggest that DAD has an important role in triggering and maintaining BVT [3,4]. In clinical situations, BVT caused by DAD can be suppressed by adenosine triphosphate, verapamil, or propranolol. However, in this case, the concentration of aconitine was high enough to render these drugs ineffective.

The use of aconitine has been known since ancient times. Clinically, it is used as an anti-inflammatory analgesic for various conditions, especially rheumatoid arthritis, sciatica, and as a cardiotonic agent. Patients with aconite poisoning typically experience symptoms within an hour, which can be as short as ten minutes after ingesting the herbs. They usually present with paraesthesia, numbness affecting the mouth and limbs, weakness, nausea, and vomiting. Ventricular tachyarrhythmias and refractory cardiovascular collapse such as in the case of this patient account for life-threatening toxicities in severe aconite poisoning [5]. While aconitine is considered to be mainly absorbed by the upper gastrointestinal tract, the most important pathway in excretion of aconitine appears to be via the kidney. The time of half-life in serum is reported to be about 6–12 h depending on renal function or cardiac output [6].

Hence prompt application of PCPS, which was continued until the aconitine was metabolized, proved successful in this case and should be considered as a management approach in cases of severe aconite poisoning.

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